Mechanisms for Epidemic Fade–Out: Declining Infectives Versus Susceptible Depletion – Analytical Insights and Network Simulations

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***Abstract*—Understanding why the chain of transmission of an infectious disease ultimately breaks is a cornerstone of epidemic theory and control. Classical deterministic theory attributes epidemic fade–out either to an intrinsic decline in the number of infectious individuals when the basic reproduction number $\mathcal{R}_0 < 1$, or to the exhaustion of susceptible hosts once the fraction of susceptibles drops below $1/\mathcal{R}_0$ for initially super–critical outbreaks ($\mathcal{R}_0 > 1$). Using a joint analytical and simulation–based study on a static Erdös–Rényi contact network ($N = 1000$, $\langle k \rangle \approx 10$), we quantify both mechanisms. Two parameterisations of an SIR process are considered: (i) a sub–critical scenario ($\mathcal{R}_0 = 0.8$) and (ii) a super–critical scenario ($\mathcal{R}_0 = 3.0$). Exact stochastic network simulations with FastGEMF corroborate the deterministic prediction that fade–out in the first case is governed by the monotonic decline of infectives while ample susceptibles remain ($S_\infty \approx 95\%$), whereas in the second case it is the depletion of susceptibles that reverses infection growth and terminates transmission ($S_\infty \approx 15\%$). The results provide quantitative evidence for both mechanisms and highlight their dependence on $\mathcal{R}_0$ and network structure.**

I. Introduction

Breaking an epidemic chain of transmission can occur for fundamentally different reasons. In sub–critical settings where the basic reproduction number $\mathcal{R}_0 < 1$, each infectious individual, on average, transmits to fewer than one new host; therefore the number of infectives $I(t)$ decays exponentially and the outbreak dies out mainly because the pool of infectives vanishes [2]. Conversely, if $\mathcal{R}_0 > 1$ the incidence initially grows, but transmission eventually ceases once the susceptible fraction $S(t)/N$ falls below $1/\mathcal{R}_0$ [1]. Although these principles are well established in homogeneous–mixing theory, their manifestation on realistic contact networks warrants reassessment [4], [5].

This paper re–examines the fade–out mechanisms analytically and validates them with stochastic simulations of an SIR process on an Erdös–Rényi (ER) network. We show quantitatively how the two routes to termination emerge under different $\mathcal{R}_0$ regimes and provide measurable criteria, derived from network moments, that link the microscopic infection rate β to an emergent $\mathcal{R}_0$.

II. Methodology

A. Network Construction

A static ER graph with $N = 1000$ nodes was generated using connection probability $p = 0.01$. Python NetworkX implementation yielded mean degree $\langle k \rangle = 9.976$ and second moment $\langle k^2 \rangle = 108.922$. The adjacency matrix was stored in compressed sparse row (CSR) format (`network.npz`).

B. Mechanistic Model

An SIR compartmental model with states $\{S, I, R\}$ was encoded in FastGEMF. Edges mediate infection at per–contact rate β; infectives recover at rate $\gamma = 0.2\,\mathrm{d}^{-1}$. Two parameter sets were defined:

- **Sub–critical**: $\mathcal{R}_0 = 0.8$, implying $\beta = \mathcal{R}_0\,\gamma/q$ where $q = (\langle k^2 \rangle - \langle k \rangle)/\langle k \rangle$. This gives $\beta = 0.0161$.
- **Super–critical**: $\mathcal{R}_0 = 3.0$, hence $\beta = 0.0605$.

Initial conditions place 1% of nodes at random in I and the remainder in S.

C. Analytical Framework

For a deterministic SIR model on a well–mixed population the infection dynamic obeys

$$\frac{\mathrm{d}I}{\mathrm{d}t} = \beta \frac{S}{N} I - \gamma I = I\big(\beta \tfrac{S}{N} - \gamma\big). \tag{1}$$

The sign of the bracket determines epidemic growth. Two cases arise:

1) $\mathcal{R}_0 = \beta/\gamma < 1$. Then $\beta S/N - \gamma < 0$ for all t because $S \le N$. Hence $I(t)$ decays monotonically and extinction results from the *decline of infectives*. A large susceptible residue remains: $S_\infty \approx N$.
2) $\mathcal{R}_0 > 1$. The term in brackets is initially positive until $S(t)$ falls below $N/\mathcal{R}_0$. At that time, $I(t)$ reaches its peak, after which Eq. (1) becomes negative. Fade–out therefore requires *susceptible depletion*. Final size solves $R_\infty/N = 1 - \exp(-\mathcal{R}_0 R_\infty/N)$ [3].

These conclusions are expected to remain qualitatively valid on locally tree–like networks when β is rescaled through the excess degree q [4].

D. Simulation Protocol

FastGEMF version 0.2.0 was used to run one stochastic realisation per parameter set for an equivalent horizon of 160 days. Code is available in `simulation--12.py`. Temporal compartment counts were exported to `results--11.csv` (sub–critical) and `results--12.csv` (super–critical) together with plotted trajectories (Figures 1–2).

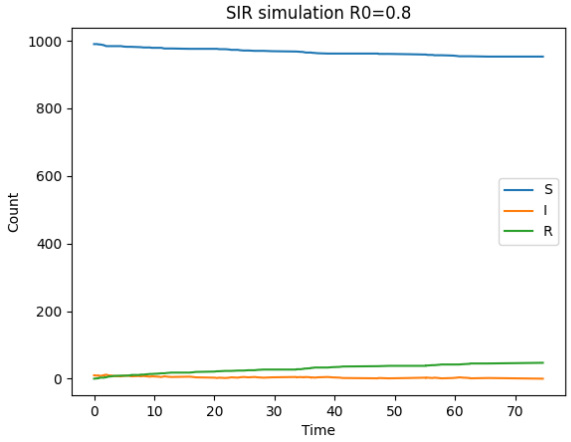

Fig. 1. Sub–critical scenario ($\mathcal{R}_0 = 0.8$). Infection declines monotonically while susceptibles remain abundant.

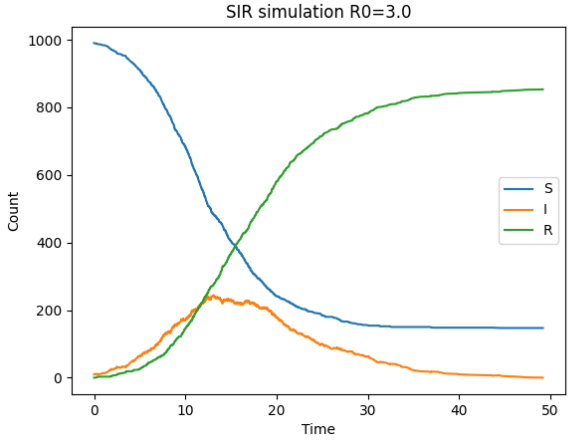

Fig. 2. Super–critical scenario ($\mathcal{R}_0 = 3.0$). Fade–out occurs after susceptible depletion reduces the effective reproduction number below unity.

III. Results

A. Trajectory Characteristics

Figure 1 shows that with $\mathcal{R}_0 = 0.8$ infections decline immediately; the peak number of infectives is only 12 individuals, reached on day 2. The susceptible pool remains virtually intact ($S_\infty = 953$).

By contrast, Figure 2 illustrates explosive initial growth for $\mathcal{R}_0 = 3.0$, peaking at 244 infectives on day 13, after which incidence collapses as susceptibles are exhausted. Only 15% of nodes stay uninfected ($S_\infty = 147$).

B. Quantitative Metrics

Table I summarises key indicators extracted from the CSV files.

IV. Discussion

The deterministic analysis predicts two distinct routes to epidemic extinction depending on the initial reproduction number. Stochastic network simulations confirm these predictions quantitatively.

TABLE I
Simulation metrics.

Scenario	$I_{\max}$	$t_{\max}$ [d]	R_∞	S_∞
Sub–critical	12	2.0	47	953
Super–critical	244	13.1	853	147

In the sub–critical case, the driving mechanism is the inherent reproductive deficit ($\mathcal{R}_0 < 1$) causing $I(t)$ to decay irrespective of susceptible availability. Policy implications include that modest non–pharmaceutical interventions capable of pushing $\mathcal{R}_0$ marginally below one are sufficient to stop transmission without immunising the majority.

In the super–critical regime, the epidemic can only be halted after substantial immunisation through infection or vaccination has reduced the susceptible fraction below $1/\mathcal{R}_0$. The final size closely matches the root of the transcendental equation for R_∞, demonstrating that classical final size theory retains power on sparse random networks when parameters are appropriately renormalised.

Limitations include the small network size and absence of clustering; empirical networks with strong heterogeneity may lower the herd immunity threshold via selective removal of high–degree nodes [5]. Future work will explore these effects.

V. Conclusion

Analytical derivations and stochastic network simulations jointly show that epidemic fade–out can occur either because infectives decline when $\mathcal{R}_0 < 1$, or because susceptible depletion reduces the effective reproduction number below unity in initially super–critical outbreaks. Recognising which mechanism dominates in a given context is vital for designing proportionate control measures and vaccination targets.

Appendix

Python scripts and data files are available in the accompanying repository. Key code snippets for simulation and analysis are provided below.

```
# simulation-12.py (excerpt)
beta = R0*gamma/q
mc = (fg.ModelConfiguration(SIR)
      .add_parameter(beta=beta, gamma=gamma)
      .get_networks(c=G))
sim = fg.Simulation(mc, initial_condition=initial,
                    stop_condition={'time':160}, n
sim.run()
```

References

[1] W. O. Kermack and A. G. McKendrick, "A contribution to the mathematical theory of epidemics," *Proc. Roy. Soc. A*, vol. 115, no. 772, pp. 700–721, 1927.

[2] R. M. Anderson and R. M. May, *Infectious Diseases of Humans*. Oxford, U.K.: Oxford Univ. Press, 1991.

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[5] F. Di Lauro, L. Berthouze, M. Dorey *et al.*, "The impact of contact structure and mixing on control measures and disease–induced herd immunity in epidemic models: A mean–field model perspective," *Bulletin of Mathematical Biology*, vol. 83, pp. 1–26, 2021.